

A CASE OF CHRONIC PROGRESSIVE OPHTHALMOPLÉGIA.

By Dr. Isador Abrahamson.

The patient was a man, twenty-seven years of age; single; a butcher by occupation, and a native of Germany. His family history was negative, with the exception of the fact that his mother had been paralyzed for years. The patient was anemic as a child, and had diphtheria when he was twelve years old. He smoked much, and drank beer excessively in former years. Eight months ago he had a small ulcer on the penis, which left no scar. He had no secondary symptoms of syphilis. His eyes were apparently normal up to six months ago, the date of the onset of his present illness. At that time he complained of pain in the left knee, like rheumatic pains. There was also general formication, and he was easily fatigued. Soon afterwards, there was pain in the left upper extremity, and immobility of the eyes. He could not rotate them, and the eyelids could not be raised. His speech was slightly altered and he felt tired when he spoke for a time, although his voice remained unchanged in pitch, etc. There was slight difficulty in swallowing. He also complained of delay in urination, with diminished potency, and constipation.

A physical examination showed a ptosis of both eyes; the pupils were equal, and reacted normally. The ocular movements were very much limited from side to side; also upwards, and to a lesser extent downwards. Convergence was preserved; divergence was considerably diminished. The superior oblique muscle seemed to be least involved. The lines on the right side of the face were flattened, and there was some weakness on that side. The mouth was drawn somewhat to the left, and hearing and bone conduction were diminished on the right side. There was weakness of the pharyngeal musculature. The tongue was protruded straight; there was no tremor; motor power in the upper and lower extremities was normal. There were no fibrillary twitchings; Romberg absent; knee jerks lively; Achilles lively; no clonus; sensibility normal; electrical examination negative. The disease was rather sudden in its onset, but made very little progress. Specific treatment and little or no influence up to the present time.

Clinically, Dr. Abrahamson said, the condition was a progressive chronic ophthalmoplegia. Whether it was of luetic nature, or indicated the early stages of a high tabes the future could only decide, yet the integrity of the pupils in spite of the extensive involvement of the ocular muscles seemed to negative the latter.

A CASE OF CONGENITAL MYOTONIA (THOMSEN'S DISEASE)
ASSOCIATED WITH OPHTHALMIC MIGRAINE.

By Dr. Charles E. Atwood.

The patient was a Scotchman, twenty-three years old; single; a book-keeper by occupation. He complained of left-sided headache, with left ptosis; also stiffness and weakness of the voluntary muscles, especially after rest, and becoming better with use. One sister, aged thirty-five, has had the same muscular disorder since childhood, but to a less marked degree than the patient. His oldest brother, when about ten years old, "could not start quickly to run." Otherwise, the family history was negative.

The patient, as a child, had pneumonia and incontinence of urine. At

nine years he had little strength to stand, and the wind from a passing football knocked him down. At the age of eleven or twelve he noticed that he went upstairs slowly, and could not lift heavy things. He also began to suffer from attacks of migraine on the left side, with vomiting, etc., occurring every six or eight weeks. He was also very constipated. At seventeen, the left eyelid began to droop during these attacks. When at play, he noticed that after resting it would be hard for him to start again, and he began to fear to go into a crowd because he could not move quickly enough to protect himself. These symptoms had persisted since, the migraine occurring intermittently for a week at a time about every six weeks, and when the patient was first seen at the Vanderbilt clinic he was suffering from one of these attacks of migraine, associated with paresis of the left third nerve, which disappeared in a few days. Other cranial nerves were free. The eye muscles affected were the levator palpebræ, the left superior and inferior recti, and the inferior oblique.

The patient was well formed, and showed excellent development of muscles, without local hypertrophies or other external defects, the strength not being proportionately great. After rest, the voluntary muscles showed stiffness and rigidity or spasticity, both flexors and extensors and other groups being affected. The condition gradually wore off with exercise of the various muscle groups, only to recur again after a period of rest. There were no sensory changes to heat, cold, pain or touch. The right knee jerk was normal; the left diminished. He showed plainly the well-known myotonic (persisting) reaction of muscles to percussion and also the Erb (persisting) reaction after prolonged faradic stimulation and removal of the electrodes. Co-ordination and position and muscle sense were apparently unaffected. There was no Romberg; no astereognosis. There was no ankle clonus or Babinski. The patient was not paralyzed at all, but there was a feeling of resistance or persistent contraction which he had to overcome in performing any act. Mental excitement exaggerated the stiffness of the muscles. Ergograph tracings by Dr. Scripture showed an incomplete relaxation of both flexors and extensors at the initial of the muscle movement. Electric stimulation of the abductor indicis showed the same thing.

Dr. George W. Jacoby said he believed he was the first one in this country to demonstrate a case of congenital myotonia, and in connection with that disorder he thought it well to emphasize a little more strongly the myotonic reaction, because after all the diagnosis would depend upon that. In the case shown by Dr. Atwood, there was apparently no question in regard to the correctness of the diagnosis, although the history was a complicated one and contained some reference to a cerebral trouble. The case would probably have to be classed in Erb's second group, in which it was questionable whether the muscular disorder was primary or secondary.

In a typical case of myotonia, the three cardinal signs upon which to base the diagnosis and which constituted the myotonic reaction, were the persistence of the muscular contractions to the faradic stimuli, the same persistence to mechanical excitation, and the persistent contracted state of muscular groups after voluntary action.

Dr. Clark said that in looking over the literature of myotonia, he was surprised to see that hypertrophy was so little dwelt upon by Hale White, Thomsen and others. Writers agreed that at most the muscle state could easily be embraced in calling it the athletic type.

The possibility that the case shown by Dr. Atwood was not of the

congenital type was worthy of considerable attention, in view of the fact that the patient had suffered from paroxysmal migraine and constipation for many years. Possibly, the muscle state here was of toxic origin, which also had some bearing on typical Thomsen's disease, as had been demonstrated by Goldflam and others for family periodic paralysis.

Dr. Terriberry said he had reported two cases of myotonia occurring in the same family, both girls. In neither of them was there noticeable hypertrophy of the muscles. He was inclined to think that hypertrophy in these cases was a less marked feature in females than in males.

ANTERIOR POLIOMYELITIS WITH INCREASED KNEE JERK.

By Dr. Atwood.

The patient was a girl, three years old, who had enjoyed good health and had been able to walk at the age of two years. Last June, *i. e.*, six months ago, she had an attack of fever lasting two days, following which she has limped slightly in the right leg. The muscles permanently affected are the posterior tibial. In these there is reaction of degeneration. The knee jerk of the affected limb is greater than that of the left, and the case is interesting on this account.

A CASE OF ACUTE POLIOMYELITIS IN AN ADULT.

By Dr. Atwood.

The patient was a man, thirty-three years old; a college student. His family and previous history were negative.

In August, 1906, nearly five months ago, he had a fever of several days' duration, accompanied by certain subjective symptoms, and the gradual onset of paralysis in his right arm, beginning in the shoulder and upper arm and culminating in the hand and finger muscles after thirty hours. An examination showed that sensation to heat and cold, pin prick and cotton were preserved. There was marked wasting of the right upper arm, shoulder and extensor surface of the forearm; also slight wasting of the right anterior tibial. The hand and affected portions of the arm were cooler than the left arm to the touch. The position of the hand was peculiar, on account of the extensor paresis. The muscles affected were the pectoralis major (upper portion), the supra and infra-spinatus, the deltoid and coraco-brachialis, the biceps, triceps, the extensors of the wrist and the long extensors of the fingers. There was also weakness of the pronators and supinators, and of the abductor pollicis.

This case, Dr. Atwood said, was thought to be of some interest on account of the comparative rarity of acute anterior poliomyelitis in the adult, and of the clearness of the subjective symptoms. In connection with the treatment of this case, the speaker inquired regarding the advisability of nerve transplantation.

Dr. Leszynsky, in discussing Dr. Atwood's first case of poliomyelitis, with increased knee jerks, said he had seen a number of cases in children where in the early stage of the disease the knee jerk was entirely absent on the affected side, but gradually returned and remained active, while the peroneal group of muscles was paralyzed and remained so.

Dr. Clark said it had occurred to him that we had a very simple explanation for the exaggeration of the knee jerks in certain types of poliomyelitis; *i. e.*, those affecting the lower posterior group of muscles